MEDICAL ASPECTS OF COLD WATER IMMERSION, A REVIEW

William A. Tansey

Naval Submarine Medical Research Laboratory
Groton, Connecticut

19 September 1973

UNCLASSIFIED
Security Classification

DOCUMENT CONTROL DATA - R & D

Security classification of title, body of abstract and indexing annotation must be entered when the overall report is classified)

1 ORIGINATING ACTIVITY (Corporate author) NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY Naval Submarine Medical Center	2a. REPORT SECURITY CLASSIFICATION Unclassified 2b. GROUP N/A

3 REPORT TITLE

MEDICAL ASPECTS OF COLD WATER IMMERSION, A REVIEW

4 DESCRIPTIVE NOTES (Type of report and inclusive dates)

Review report

5 AUTHOR(S) (First name, middle initial, last name)

William A. Tansey, LT, MC, USNR

6 REPORT DATE 19 September 1973	7a. TOTAL NO. OF PAGES 15	7b. NO. OF REFS 81
8a CONTRACT OR GRANT NO b. PROJECT NO MF51.524.006-9025A9K c. d.	9a. ORIGINATOR'S REPORT NUMBER(S) NavSubMedRschLab Report No. 763 9b. OTHER REPORT NO(S) (Any other numbers that may be assigned this report)	

10 DISTRIBUTION STATEMENT

Approved for public release; distribution unlimited

11 SUPPLEMENTARY NOTES	12 SPONSORING MILITARY ACTIVITY Naval Submarine Medical Center Box 600 Naval Submarine Base Groton, Connecticut 06340

13 ABSTRACT

Whole body hypothermia is an acknowledged cause of death in victims of cold water immersion. Survivors of submarine escape must contend with this physiologic stress in the open sea. A concise statement of basic physiologic principles and modes of treatment is a timely adjunct to training programs for submarine escape so that victims may better cope with hypothermic environments.

DD FORM 1 NOV 65 1473 (PAGE 1)

S/N 0102-014-6600

Unclassified
Security Classification

14 KEY WORDS	LINK A		LINK B		LINK C	
	ROLE	WT	ROLE	WT	ROLE	WT
Hypothermia						
Cold Water Immersion						
Frostbite						
Immersion Foot						
Open Sea Survival						
Environmental Stress						

MEDICAL ASPECTS OF COLD WATER IMMERSION, A REVIEW

by

William A. Tansey, LT, MC, USNR

NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY
REPORT NUMBER 763

Bureau of Medicine and Surgery, Navy Department
Research Work Unit MF51.524.006-9025BA0K.41

Transmitted by:

Valentine David Galasyn

Valentine D. Galasyn
CDR, MC, USN
Director, Naval Undersea Medical Institute

Reviewed and Approved by:

Charles F. Gell

Charles F. Gell, M.D., D.Sc.(Med)
Scientific Director
NavSubMedRschLab

Approved and Released by:

R. L. Spiar

R. L. Spiar, CDR MC USN
Officer-in-Charge
NavSubMedRschLab

SUMMARY PAGE

THE PROBLEM

To compile a review and bibliography of available information on medical aspects of cold water immersion.

FINDINGS

The clinical pathogenesis and treatment of acute hypothermia are defined and discussed.

APPLICATION

Submarine candidates in the U.S. Navy are trained in the techniques of buoyant ascent from hyperbaric environments. More emphasis on survival at the surface of the open sea is required. This review is intended as a useful reference for medical personnel involved in instruction of escape procedures.

ADMINISTRATIVE INFORMATION

This paper was prepared by the author in partial fulfillment of the requirements for qualification as a Submarine Medical Officer. It was selected for publication by the Naval Submarine Medical Research Laboratory in order to make the material generally available in the Technical Library and as reference material in the Naval Undersea Medical Institute.

The manuscript was submitted for review on 11 June 1973, approved for publication on 19 September 1973 and designated as NSMRL Report No. 763.

PUBLISHED BY THE NAVAL SUBMARINE MEDICAL RESEARCH LABORATORY

ABSTRACT

Whole body hypothermia is an acknowledged cause of death in victims of cold water immersion. Survivors of submarine escape must contend with this physiologic stress in the open sea. This report contains a concise statement of basic physiologic principles and modes of treatment and is considered a timely adjunct to training programs for submarine escape so that victims may better cope with hypothermic environments, and medical personnel may be better able to revive victims rescued from such accidents.

MEDICAL ASPECTS OF COLD WATER IMMERSION, A REVIEW

INTRODUCTION

Accidental whole body hypothermia threatens anyone who might suddenly be immersed in cold water, either in inland waters or in the open sea. Hypothermia is probably a more common cause of death among victims of shipwreck than drowning.[58] In training submarine personnel for escape and survival, the Navy stresses the hazards of ascent from hyperbaric environments. It is felt that additional emphasis should be placed on the hazards of cold water exposure.

W. R. Keatinge's recent monograph "Survival in Cold Water" consolidates many current concepts.[37] Additional information and advice derive from several medical disciplines. Data accumulated from well controlled physiologic experiments with animals and humans, case reports of alcohol and barbiturate overdoses, documentaries of shipwreck survivors, and tales of mountain climbers overcome by extreme cold, all contribute insight into the complexities of the physiology and treatment of whole body hypothermia. Induced hypothermia has been explored in clinical medicine in an attempt to minimize metabolic demands during surgery and to arrest blood loss during gastrointestinal hemorrhage. Only the infamous experiments of a pharmacologist at Dachau have directly explored the lethal limits of human cold tolerance by cold water immersion;[19] and the uncertain physical fitness of those unanesthetized prisoner-victims detracts further from those studies.

This report is intended to afford those occupationally and recreationally exposed to hypothermic environments with a basic understanding of the effects of cold upon physiologic systems. It is hoped that it will prepare the diving and submarine medical officers to respond to the needs of the victims of cold water immersion, and that potential survivors of submarine escape will be taught to understand the hazards of the open sea. More detailed physiologic reviews, experimental data, and case reports are referenced in the bibliography.

PRINCIPLES OF HEAT CONSERVATION

Man is unable to endure precipitous falls in core temperature. A central thermoregulatory system in the hypothalamus maintains an approximately constant core temperature by integrating the cardiovascular, pulmonary, and metabolic responses to cold stress. The temperature in superficial regions varies, allowing a gradient from the body core to the body surface. Heat is continuously produced by basal oxidative metabolism. The hypothalamus responds both to the temperature of circulating blood and to the afferent impulses from thermal sensors on the skin and internal surfaces. Basal heat production can be augmented twentyfold by physical exercise and five to sixfold by shivering.[28,62] Heat exchange with the

environment is accomplished by radiation, conduction and convection, and evaporation. At equilibrium, Newton's law of cooling obtains:

Heat Production $\pm$ Exchange with Environment - Heat loss by Evaporation = 0

Convection is a major pathway for heat exchange from the body to normal air environments. The rate of heat loss by convection varies with the density of the surrounding environment and with the temperature gradient at the skin. Since cold water is denser than air and its thermal conductivity is twenty-five times that of air, a large temperature gradient at the skin-water interface can severely challenge thermal balance.

In resting man, unclothed, and immersed in water of 20-25°C, heat loss soon exceeds endogenous heat production and the core temperature falls. Where air temperature is also low, respiratory heat loss becomes increasingly significant. Decreasing core temperature impairs central regulation. At 30°C central regulation ceases and man becomes poikilothermic. Cold stress is greater at increased ambient pressure;[68] the explanations are speculative and will bear heavily upon the excursion profiles of future saturation diving programs. Respiratory heat loss accelerates with the increasing density of the breathing medium.

Death from cold is due to physiological derangements rather than to the direct effect of cold upon living tissue. In reviewing records of shipwrecks maintained by the U.S. Navy's Bureau of Medicine and Surgery, Molnar found that death due to hypothermia occurred at nearly the same temperature for all victims.[58] Survival times were a function of the rate of cooling.

The body's insulative "shell" varies with the thickness of the subcutaneous adipose tissue[10] and the conduction of heat through this tissue varies with degrees of perfusion. Almost immediate vasoconstriction, both from a central reflex and from direct effects of cold upon vascular smooth muscle, diminishes circulation and minimizes the conduction of heat to the body surface. In effect, the thickness of the insulative layer is increased. The extremities are also heat exchangers and their effect can be variably bypassed by alterations in vascular tone.

Control of peripheral circulation is lost with severe cooling. As skin temperature approaches 10-12°C, cold paralysis of the vascular smooth muscle causes vasodilatation.[37] Tissue conductance is then increased and heat loss is accelerated. This phenomenon, "cold-induced vasodilatation" (CIVD), is in part explained by the vessel walls becoming refractory to norepinephrine. Ordinary clothing can often supplement the body's insulation sufficiently to protect man from this usually lethal event.

Shivering thermogenesis can account for a five to sixfold increase in body heat production.[62] The involuntary muscular contractions of shivering generate heat by splitting Adenosine Triphosphate (ATP) to Adenosine Diphosphate (ADP) without net mechanical work. The ADP stimulates the electron transport system and oxygen con-

sumption. The increased oxidative metabolism involves fat, carbohydrate and protein as substrates.[53] Voluntary exercise, too, can increase endogenous heat production by increasing oxygen consumption; the attendant increase in vascular perfusion to the exercising muscles, however, increases tissue conductance and counteracts the conservation of heat by vasoconstriction. Both shivering and voluntary exercise are subject to fatigue.

Cellular metabolism varies in direct proportion to temperature and obeys the Van't Hoff-Arrhenius Law which states that the rate of a chemical reaction doubles or triples for each 10° C increase of temperature. The converse is true.[56] Tissue oxygen requirements are therefore diminished so that a minimal oxygen debt develops when circulation is compromised at low temperatures.

Certain aquatic people, such as the Korean Ama, who are voluntarily exposed to cold stress on a chronic basis, are acclimatized such that they are able to maintain a higher metabolic rate than their non-diving counterparts and thus endure more severe cold stress.[36] The degree of acclimatization has no major practical significance.

THE HEART

The cardiovascular system suffers the most profound changes in accidental hypothermia. There are degrees of cardiac rhythm alteration which reflect changes in core temperature. Sudden exposure to cold water, as with ice water showers,[41] will cause an increase in arterial pressure, pulse rate, cardiac output, and cardiac work. The absence of an increase in circulating adrenalin suggests direct reflex rather than secondary sympathetic response.[41] The acute increase in pulse rate is possibly dampened during water immersion by the bradycardia which accompanies submerging the face, a diving reflex mediated by the trigeminal and vagal nerves.[28] Ventricular ectopic activity is noted during the first three minutes of cold immersion but then subsides.[41] This too may be related to a cold reflex from the skin, or to the abrupt increase in cardiac work. The popular concept of reflex ventricular fibrillation has little experimental basis and, while such a reflex may occur, it is probably not a common cause of sudden death in cold water.

No single etiology for ventricular fibrillation has emerged. Controlled studies where changes in pH and P_{CO_2} have been minimized, have demonstrated that low temperature alone does not consistently lead to ventricular fibrillation. Respiratory depression and concomitant changes in P_{CO_2} and in the blood pH are more directly linked to the production of arrhythmias.[33] Rapid decreases in P_{CO_2} have been implicated in the induction of ventricular fibrillation — a caution which must be reckoned with during the emergency resuscitation of hypothermic patients.

Although oxyhemoglobin dissociation is impaired, a constancy in coronary arterovenous oxygen difference suggests that myocardial oxygen requirements are nearly met.[63] Indeed, the impairment of dissociation is thought to be

counteracted by a fall in pH. The arrhythmia, then, is probably not ischemic in origin.

Finally, an increase in the concentration of thyroid hormones in the specialized conduction fibers is known to abet ventricular excitability. Such an increase has been measured during induced hypothermia.[25]

The electrocardiogram of hypothermia is characteristic. The sinus rate slows progressively as the core temperature of the victim approaches 34°C. T-waves are inverted. As conduction is slowed, PR, QRS, and QT intervals are prolonged owing to decreased resting potential, to diminished amplitude and slow upstroke of the action potential, and to increased durations of the relative and absolute refractory periods within the conduction system.[33]

Auricular muscle tends to be more sensitive to cold than the Purkinje system of the ventricle. Near 29-31°C atrial fibrillation with a slow ventricular response is common. Ventricular excitability manifest by premature ventricular contractions frequently heralds the onset of ventricular fibrillation near 28°C. Complete standstill is expected at heart temperatures less than 20°C.

More subtle electrocardiographic signs appear in the initial phases of body cooling. While the cadaveric muscle rigidity seen near 32°C may not be accompanied by visible shivering, a fine muscle tremor artifact becomes increasingly apparent on the electrocardiogram. A slowly inscribed terminal QRS abnormality most prominent in the middle and lateral precordial leads has been designated a "J-wave" or "Osborn wave". This has been variously interpreted as representing anoxia, injury current, delayed ventricular depolarization, or an "early" repolarization in a portion of the ventricle before delayed depolarization is complete in another portion.[72] The Osborn wave is easily detected in extreme hypothermia and less easily detectible in milder degrees.

These electrocardiographic changes are reversible with rewarming suggesting that there has been little if any direct damaging effect by cold upon muscle or conduction fibers. High levels of creatine kinase and of alpha-hydroxybutyrate dehydrogenase in patients with accidental hypothermia could, nevertheless, indicate skeletal as well as cardiac muscle damage.[52] Only the prolonged Q-T interval is slow to resolve.

The electrocardiogram has considerable clinical relevance as a useful indicator of fine alterations in core temperature. Rectal temperatures may differ by as much as 2.3°C from deep core temperature,[73] and are therefore not entirely reliable. Temperature determination at the lower one third of the esophagus is a good index of the temperature of the central circulation; however, manipulation of the larynx while positioning the thermistor probe risks the induction of further reflex bradycardia and/or cardiac standstill. Ear temperatures correlate well with observed thermal changes in the central nervous system but do not reflect core temperature. Unfortunately, most readily available thermometers are only calibrated to 34°C. Suffice it is to say that the combination of atrial fibrillation with slow ventricular response, ectopic

ventricular activity, muscle tremor artifact, and a prolonged QRS complex with an Osborn wave in a victim of accidental cold water immersion is ominous. Degrees of electrocardiographic change will parallel degrees of core temperature compromise.

While the immediate response to cold is an augmentation of cardiac output and arterial pressure, both of these parameters decline with progressive cooling, more as a result of bradycardia than of diminished stroke volume. Blood pressure will be difficult to obtain near 33°C. It must be emphasized, however, that as long as a regular cardiac rhythm is maintained at low temperatures, it is probable that the oxygen requirements of critical organs are being met.

THE LUNG

Sudden exposure to ice water with a temperature of less than 25°C causes an immediate involuntary increase in the rate of respiration with an inspiratory shift. Arterial P_{O_2} increases. Keatinge observes that the reflex occurs in decerebrate cats, and argues that it is initiated by cold receptors in the skin, especially in the chest and abdomen, and is mediated at the midbrain level.[39] A subjective sense of difficulty in breathing predominates. Clearly, when one is immersed in the cold sea at the end of an expiratory phase of breathing, he risks uncontrolled aspiration of a large volume of water; drowning would be inevitable.

Hyperventilation in this sudden cold exposure, with its attendant hypocarbia and respiratory alkalosis, further impairs oxyhemoglobin dissociation. The respiratory effort impedes cardiac filling and output. Conditioned cold water swimmers enjoy relative freedom from the respiratory distress.[40]

Breathing rate falls progressively with the fall in core temperature which accompanies prolonged cold exposure. The respiratory quotient (CO_2 expired/O_2 inhaled) also declines as CO_2 solubility in plasma increases and O_2 consumption falls. While in dogs bronchodilation can lead to a 70-90% increase in anatomic dead space, and while ventilation decreases, CO_2 excretion is not impaired.[69] Pulmonary diffusing capacity as measured by carbon monoxide technique is reduced at lower temperatures probably owing both to slower kinetics of carbon monoxide with blood and to reduced membrane diffusing capacity.[62,61] Nevertheless, the reduced diffusion capacity for CO_2 and O_2 is adequate to serve diminished metabolic requirements.

Pulmonary compliance is not impaired unless gross aspiration has occurred, in which case one is dealing with near drowning rather than with pure immersion hypothermia.[57]

THE CENTRAL NERVOUS SYSTEM

The brain is highly sensitive to temperature changes because of its high level of metabolic activity. Depression of this activity with moderate degrees of hypothermia has been a useful tool for neurovascular surgeons.

Temperature limits for the human central nervous system are highly vari-

able. A phase of hyperexcitability down to 35°C precedes uniform central depression. Before becoming unconscious near 33°C, one becomes dysarthric and begins to lose contact with his surroundings. With only moderate cold stress, one develops amnesia for the period of cooling.

THE MUSCLE

Differential conduction rates in nerve and muscle are reflected in progressively disorganized muscle activity. Muscular rigidity is prominent in the early phases of severe cold stress and manual dexterity is compromised. Abrupt relaxation in severe cold usually heralds death.[19]

THE KIDNEY

Most everyone who has had the experience of swimming in cold water is familiar with the phenomenon of cold diuresis. In spite of diminished glomerular filtration rate and renal blood flow,[18,56] a diuresis occurs which can be counteracted by pitressin.[37] Keatinge postulates that this is a physiologic compensation for the relative increase in central blood volume, a result of peripheral vasoconstriction. The reduction in plasma volume becomes critically relevant during treatment.

Cold directly affects the enzymatic activity in the distal renal tubules. This is manifest in the altered resorption and secretion of certain substances;[56] for example, glycosuria may occur in the presence of normoglycemia. Blood urea nitrogen might be expected to be elevated after prolonged exposure.

THE BLOOD

Fluid shifts from the vascular space together with contraction of the plasma volume by cold diuresis leads to hemoconcentration. Blood viscosity increases markedly[19,61] lending to peripheral vascular sludging and increased cardiac work load.

Reports on clotting are at best conflicting. Silicon clotting time, two-stage prothrombin time, and bleeding time have been shown to increase.[18] ADP platelet aggregation is decreased at very low temperatures[35] and platelets show considerable change in shape.[61] Platelet physiology is altered by low temperatures but the particular physiologic events have yet to be defined.

THE ENDOCRINE SYSTEM

In vivo endocrine activity in hypothermia is difficult to assess because of the varied rates of detoxification of circulating hormones. TSH secretion is enhanced by cold, at least in part to meet the increased requirement for heat production. Circulating levels of T_3 and T_4 remain constant,[37,77] probably owing to enhanced peripheral disposal.[17] Chronic cold exposure stimulates the pituitary-adrenal axis; the moderately increased corticosteroid output potentiates epinephrine in cold-induced fat mobilization and both epinephrine and corticosteroid activate

the oxidation of glucose — two substrates for nonshivering thermogenesis. Controlled observations detected no acute changes in growth hormone levels induced by cold.[22]

PERIPHERAL COLD INJURY

Local cooling of muscle favors the onset of cramps. These are related to delayed breakdown of acetylcholine and to slowing of the motor endplate repolarization.[37] With extended exposure to cold but not freezing water, prolonged peripheral vasoconstriction and hyperviscosity can produce local ischemic changes in muscle and nerve tissue. The so-called "immersion hand" or foot presents initially numb, pale and pulseless; with judicious rewarming, full pulses return to a hyperemic, painful, swollen, and partially or fully paralyzed limb; weeks later, a near normal post-hyperemic limb is hypersensitive to cold. Pathology in this latter stage reveals muscular and neuronal degeneration most probably from ischemia. In severe cases complete cessation of blood flow predisposes to gangrene. In treating, Behnke warns that too rapid rewarming of an immersion foot or hand could increase metabolic demands for oxygen more rapidly than the circulation can be restored, thus aggravating anoxic damage.[5] Only in the setting of whole body hypothermia could this risk be obviated by the urgent need for whole body warmth. Hence, where practical, one would submerge the trunk in water of 42-45°C degrees and allow the limbs to be rewarmed gradually from the warmer blood of the central circulation.

The true freezing point of finger tissue is -0.53°C.[37] Any person exposed to cold water in this range is not likely to be salvageable. Nevertheless, frostbite is encountered in exposed extremities; blood vessels are irreversibly injured and local circulatory arrest is complete. As interstitial fluid freezes, water is drawn osmotically out of the cells leaving destructively high intracellular electrolyte concentrations. In cases of frostbite, rapid rewarming is most effective in resolving the electrolyte imbalance and in minimizing tissue destruction.[42] As the affected extremity is rewarmed, plasma loss accelerates through damaged vessel walls. This produces local hemoconcentration; vessel blockage by sludging red cells contributes to further ischemic tissue damage. Behnke and Brauer suggest that low molecular weight dextran infusion might alleviate much of the erythrocyte aggregation,[5] but more controlled studies are indicated. Permanent tissue damage is inevitable with frostbite and the victim is likely to require chronic analgesia and/or sympathectomy.

The distinction between frost bite and immersion foot should be emphasized since the treatment differs. Frostbite is associated with acute exposures to environmental temperatures near 0°C. The formation of ice crystals damages cells both by mechanical trauma and by aforementioned osmotic gradients. Damaged capillaries exude fluid. Immersion injury results from prolonged exposure to cold but not freezing temperatures. Shipwreck survivors onboard life rafts awaiting rescue

are particularly vulnerable. Capillary sludging and thrombosis precipitate ischemic change. With frostbite, the skin is frozen while the muscle remains normal. With classical immersion injury, much necrosis and nerve damage progress under a relatively intact skin. There is surprisingly little final tissue loss in the former while the latter may be followed by permanent muscle contractures and nerve lesions.[81]

For reasons already outlined, frostbite should be treated with rapid rewarming at temperatures between 42 and 45°C. Temperatures less than 42°C achieve less salvage of tissue, while temperatures greater than 45°C compound tissue damage.[60] Treatment should be deferred until proper temperatures can be achieved. In view of the paucity of available data, slow rewarming is advocated for immersion injury, lest ischemic changes be aggravated. With a combination of frostbite and immersion injury, the indications for rapid rewarming should prevail.

It must be stressed that freezing injury and frostbite can be avoided by victims of accidental cold water immersion by keeping the extremities covered with conventional clothing (e.g. socks and mittens) which can maintain skin temperatures 4-5°C degrees above that of the surrounding water. The implications for the design of submarine escape suits are obvious.

TREATMENT OF WHOLE BODY HYPOTHERMIA

A slight increase in core temperature follows acute cold water immersion. This is followed by a slow decline to 35°C and then a rapid fall until the temperature gradient at the body-water interface is minimized. Shivering is maximum at a body temperature of 35°C and then decreases through 34°C, 33°C, 32°C and 31°C. Below the 33-31°C range shivering is replaced by a tonic stage of muscle contraction and consciousness becomes clouded; supraventricular arrhythmias appear. Near 30° 30°C these changes are complete and the subject's survival is dependent upon external aid. Below 30°C ventricular arrhythmias are expected; the subject is in grave danger. Death is most likely to ensue near 24°C.[21,28] This temperature profile is generally applicable, however, there is marked individual variation.

Once a pale, pulseless, semiconscious or unconscious individual has been retrieved from cold water, swift efforts must be made to assess the victim's core temperature with available thermistor probes, with an electrocardiogram, and with clinical estimates according to the above guidelines. General supportive care with particular attention to the maintenance of an airway must be initiated immediately. While Keatinge and Behnke urge rapid restoration of normal core temperature, Hillman advocates slow rewarming.[29,31] The rate of restoration of core temperature must be carefully balanced between the risk of anoxic damage from too brisk an increase in tissue oxygen requirement before circulation is improved and the risk of vascular collapse from the critical after-drop in core temperature associated with the restoration of peripheral blood through cold deeper layers of subcutaneous tissue. The latter risk considerably out-

weighs the former. Suggested treatment techniques include baths,[5,37] extracorporeal circulation,[15] warm air inhalation,[76] warm peritoneal dialysis,[41] and heating blankets and cradles.[47] An infant is best rewarmed by gastric lavage with a heated glucose solution via a nasogastric tube.[80] An adult, on the other hand, is best submerged in a bath of 42-45°C and kept there until his core temperature rises above 33°C at which time he might be allowed to continue to warm spontaneously. In the event that this immersion is impractical because of major injury or because of the exigencies of a cardiopulmonary arrest, the alternative modes of rewarming are to be considered with careful vigilance for the after-drop in blood pressure.

Slow rewarming becomes appropriate with prolonged cold exposure (greater than 6 hours) where hemoconcentration from fluid shifts, increased blood viscosity, exhaustion of glycogen stores, and acidosis all contribute to an upset "milieu interieur" which will be aggravated by rapid rewarming and which will revive spontaneously if further cold is prevented.[21]

In view of depressed respiration and probably atelectasis with aspiration or anoxic pulmonary edema, oxygen supplement (95%) and cautious pulmonary toilet with endotracheal suction and intermittent positive pressure breathing should be initiated. The respiratory rate should be at least half normal, lest too rapid a drop in P_{CO_2} with relative hyperventilation precipitate ventricular fibrillation. Endotracheal intubation, while greatly facilitating respiratory support in the semi- or unconscious victim is best avoided until absolutely necessary, again, because of the potential reflex bradycardia which predisposes to lethal arrhythmias. Electrocardioversion, if required, is not effective below 28°C.

Carbon dioxide and oxygen tensions should be closely monitored with careful correction for temperature and pH related shifts in the respective dissociation curves. A 1°C fall in the temperature of blood sealed in an anaerobic environment will lower the CO_2 tension 4.4% and the O_2 tension 6%.[1] Correction factors have been provided by Severinghaus.[70] Note that at 30°C an apparent P_{O_2} of 120 mmHg at a pH of 7.6 would be corrected to 70.8 mmHg.

Hemoglobin and hematocrit values may be misleading. While one expects a hemoconcentration both from contraction of plasma volume and from fluid shifts, hemodilution can occur with blood loss, with parenteral fluids, or with aspiration of hypotonic water. Blood electrolytes must also be interpreted with cognizance of these fluid shifts. Sodium bicarbonate is indicated for metabolic acidosis.

Prophylactic antibiotics are not indicated in the initial phases of treatment. The common complication of pulmonary infection is of course to be treated aggressively with antibiotics. Steroid therapy is reserved for cases of obvious aspiration. Antifibrillatory drugs are of little use and are in fact contraindicated in hypothermic states. Antihistamines are appropriate for the infrequent cold allergy.

Local injury and the rationale for therapy have been discussed in a separate section.

SUDDEN DEATH

The phenomenon of sudden death in cold water has perplexed medical officers and coroners alike. W. R. Keatinge observed that a clothed champion swimmer could last only 70 seconds swimming actively in 4.7°C water.[42] The swimmer was unable to grab the side of the pool and dropped toward the bottom like a stone. Others lasted up to twelve minutes. None of the volunteer swimmers sustained a fall in core temperature, but all collapsed from exhaustion probably owing to the high viscosity of the cold water, to the increasing work needed to produce swimming movements, and to the respiratory drive from the cold. Fatter people fared better, perhaps because of their higher buoyancy. The warning is clear to the day-sailor who capsizes in cold water 100 yards off shore.

CONCLUSION

The awesome quest for survival in the sea has always challenged man. Hypothermia is not the least of the limitations. One is afforded some protection by wearing at least normal clothing. Even while wet and cumbersome, clothing, by air trapping, can contribute significantly enough to body insulation to prevent critical excursions of core and skin temperatures. Keatinge has shown that volunteers held in 5°C water for twenty minutes wearing woolen underwear, submarine sweater, trousers and socks sustained one fourth the fall in temperature they sustained when nude.[42]

Exercise may augment thermogenesis in water above 25°C, while as noted above, in colder water, the attendant increase in tissue conduction will augment heat loss.

Alcohol induces vasodilatation. The effect upon heat loss is negligible. Indeed, alcohol can improve one's subjective response to cold with the hazard of dampening one's own sense of critical heat loss.

Fatter people tend to endure cold stress for longer periods than their more lean compatriots. This relates to greater insulation and to greater buoyancy — water density increases as it becomes colder so that a body of relatively low specific gravity is even better able to float. Nevertheless, inflatable life vests are essential for survival since whole body heat loss and consequent critical hypothermia are inevitable with prolonged cold exposure.

Proper water temperatures for treatment can be achieved in remote settings even without sophisticated temperature gauges. J. D. Nelms reports from Army Field Trials that a simple temperature sensation scale is a practical and effective means of maintaining bath temperatures between 42°C and 45°C:[60]

less than 43° C - Warm

43°C - Comfortably hot.

44°C - Maximally hot for comfort.

46°C - Definite discomfort or pain.

greater than

47°C - Quite a lot of pain.

Many victims of occupational or recreational aquatic accidents who have died from the hypothermia induced by cold water immersion might have endured longer with proper precautions. An escapee from a submarine is well versed in the technique of buoyant ascent; but will he survive on the surface? While he successfully escapes the horror of a distressed submersible he soon becomes a prisoner of the open sea. Survival then depends upon adequate equipment and proper training. These are the responsibilities of the cognizant medical authorities.

REFERENCES

1. Adolph, E. F. Effects of low body temperature on tissue oxygen utilization. The Physiology of Induced Hypothermia, Publication 451, National Academy of Sciences, National Research Council, Washington, D.C., 1956, Dripps, R. D., Editor.

2. Angelakos, E. T., Maher, J. T. and Burlington, R. F. Spontaneous cardiac activity at low temperature in hibernators and non hibernators, influence of potassium and catecholamines. Fe Pro 28, 1216, 1969.

3. Bright, C. V. Diving in the Arctic. Nav Rsch Rev 25, 1, 1972.

4. Bradley, A. F., Stupfel, M. and Severinghaus, J. W. Effect of temperature on P_{CO_2} and P_{O_2} of blood in vitro. J Appl Physiol 9, 201, 1956.

5. Behnke, A.R. and Brauer, R.W. In Harrison's Principles of Internal Medicine. 6th edition, McGraw Hill Book Company, New York, 1970.

6. Behnke, A. R. Yaglou, C. P. Physiological responses of men to chilling in ice water and to slow and fast rewarming. J Appl Physiol 3, 591, 1950.

7. Berkley, J. S. Treatment after exposure to cold. Lancet 1, 378, 1972.

8. Brooks, C. McC. Hypothermia and the nervous system. Physiology of Induced Hypothermia, Publication 451, National Academy of Sciences, National Research Council, Washington, D.C., 1956, Dripps, R. D., Editor.

9. Brooks, Chandler McC. Hypothermia and the physiology of cardiac excitability. The Physiology of Induced Hypothermia, Publication 451, National Academy of Sciences, National Research Council, Washington, D.C., 1956, Dripps, R. D., Editor.

10. Bullard, R. W. and Rapp, G. M. Problems of body heat loss in water immersion. Aerospace Med 41, 1269, 1970.

11. Clements, S. D. and Hurst, J. W. Diagnostic value of ECG abnormalities observed in subjects accidentally exposed to cold. Am J Cardiol 29, 729, 1972.

12. Costill, D. L., Cahill, P. J. and Duane, E. Metabolic responses to

submaximal exercise in three water temperatures. J Appl Physiol 22, 628, 1967.

13. Craig, A. B. and Dvurak, M. Thermal regulation of man exercising during water immersion. J Appl Physiol 25, 28, 1968.

14. Daigliesh, D. G. Cold/wet exposure ashore. J R Nav Med Serv 58, 177, 1972.

15. Davies, D. M. Accidental hypothermia treated by extracorporeal blood-warming. Lancet 1, 1036, 1967.

16. Feldman, S. A. Profound hypothermia. Br J Anasth 43, 244, 1971.

17. Fisher, D. A. and Odell, W. D. Effect of cold or TSH secretion in man. J Clin Endocrinol Metab 33, 859, 1970.

18. Fruehan, A. E. Accidental hypothermia. Arch Intern Med 106, 218, 1960.

19. Gagge, A. P. and Herrington, L. P. Physiological effects of heat and cold. Ann Rev Physiol 9, 409, 1947.

20. Golden, F. St. C. Cold water immersion. J R Nav Med Serv 58, 195, 1972.

21. Golden, F. St. C. Accidental hypothermia. J R Nav Med Serv 58, 196, 1972.

22. Golstein-Golaire, J., Vanllaelst, L., Bruno, O. D., LeClercq, R. and Copinschi, G. Acute effects of cold on blood levels of growth hormone, cortisol and thyrotropin in man. J Appl Physiol 29, 622, 1970.

23. Gregory, R. T. and Patton, J. F. Treatment after exposure to cold. Lancet 1, 377, 1972.

24. Hanna, J. M. and Hong, S. K. Critical water temperature and effective insulation in SCUBA divers in Hawaii. J Appl Physiol 33, 770, 1972.

25. Harland, W. A., Orr, J. S., Dunnigan, M. G. and Fyfe, T. Cardiac effects of cold induced hyperthroidism and seasonal variation in incidence of myocardial infarction. Br J Exp Pathol 52, 147, 1970.

26. Hegenauer, A. H., Flynn, J. and D'Amato, H. Cardiac physiology in the dog during rewarming from deep deep hypothermia. Am J Physiol 167, 69, 1951.

27. Hegenauer, A. H. and Covino, B. G. Myocardial irritability in experimental immersion hypothermia. The Physiology of Induced Hypothermia. Publication 451, National Academy of Sciences, National Research Council, Washington, D.C., 1956, Dripps, R. D., Editor.

28. Hervey, G. R. The physiology of cold/wet survival. J R Nav Med Serv 58, 161, 1972.

29. Hillman, H. Treatment after exposure to cold. Lancet 1, 378, 1972.

30. Hillman, H. Treatment after exposure to cold. Lancet 2, 1257, 1971.

31. Hillman, H. Treatment after exposure to cold. Lancet 1, 140, 1972.

32. Hoff, H. E. and Stansfield, H. Ventricular fibrillation induced by cold. Am Heart J 38, 193, 1949.

33. Hoffman, B. F. Temperature effects of cardiac transmembrane potentials. The Physiology of Induced Hypothermia, Publication 451, National Academy of Sciences, National Research Council, Washington, D.C., 1956, Dripps, R. D., Editor.

34. Jesser, K and Hagelsten, J. O. Search and rescue service in Denmark with special reference to accidental hypothermia. Aerospace Med 43, 787, 1972.

35. Kaltlove, H. and Alexander, B. Effect of cold on bleeding. Lancet 2, 1359, 1970.

36. Kang, B. S., Song, S. H., Sah, C. S. and Hong, S. K. Changes in body temperature and basal metabolic rate of the AMA. J Appl Physiol 18, 487, 1963.

37. Keatinge, W. R. Survival in Cold Water. Blackwell Scientific Publications, Oxford, England, 1969.

38. Keatinge, W. R., et al. Sudden failure of swimming in cold water. Br Med J 1, 480, 1969.

39. Keatinge, W. R. and Nadel, J. A. Immediate respiratory response to sudden cooling of the skin. J Appl Physiol 20, 65, 1965.

40. Keatinge, W. R. Swimming in cold water. Brit Med J 2, 185, 1969.

41. Keatinge, W. R. Thermal Problems in Submarine Escape. International Workshop on Escape and Survival from Submersibles. NavSubMedRschLab, Groton, Ct., June 1972 (in preparation).

42. Keatinge, W. R. Cold immersion and swimming. J R Nav Med Serv 58, 171, 1972.

43. Keen, G. and Dowlatshaki, K. The effects of circulatory arrest during profound hypothermia upon human myocardial fine structure. Cardiovasc Res 4, 348, 1970.

44. Lash, R. F., Bardette, J. A. and Osdil, T. Accidental profound hypothermia and barbiturate intoxication. A report of rapid "Core" rewarming by peritoneal dialysis. J Am Med Assoc 201, 169, 1967.

45. Laufman, H. Profound accidental hypothermia. J Am Med Assoc 147, 1201, 1951.

46. Ledingham, I. McA. Severe hypothermia with barbiturate intoxication. Lancet 1, 24, 1966.

47. Ledingham, I. McA. and Mone, J. G. Treatment after exposure to cold. Lancet 1, 534, 1972.

48. Leon, D. F., Amidi, M. and Leonard, J. J. Left heart work and temperature responses to cold exposure in man. Am J Cardiol 26, 38, 1970.

49. Lloyd, L. L. Treatment after exposure to cold. Lancet 1, 491, 1972.

50. Lloyd, L. L. Treatment after exposure to cold. Lancet 2, 1376, 1971.

51. Mackay, D. E. The problem of cold/wet survival. J R Nav Med Serv 58, 158, 1972.

52. Maclean, D. Serum enzymes in relation to ECG changes in accidental hypothermia. Lancet 2, 1266, 1968.

53. Masoro, E. J. Factors influencing intermediary metabolism. Physiology and Biophysics, 1070, Ruch, T. C. and Patton, H. D., editors. W.B. Saunders Co., Philadelphia and London, 1965.

54. McQueen, J.D. Effect of cold upon the nervous system. Physiology of Induced Hypothermia, Publication 451, Ntl Acad Sci, NRC, Wash, D.C. 1956 Dripps, R.D., Ed. p. 243.

55. Mears, G. Treatment after exposure to cold. Lancet 1, 38, 1972.

56. Meriwether, W. D. and Goodman, R. M. Severe accidental hypothermia with survival after rapid rewarming. Am J Med 53, 505, 1972.

57. Modell, J. H. Pathophysiology and Treatment of Drowning and Near Drowning. Charles Thomas, publisher, Springfield, Illinois, 1971.

58. Molnar, G. W. Survival of hypothermia by mean immersed in the ocean. J Am Med Assoc 131, 1046, 1946.

59. Munday, K. and Noble, A. R. Renin secretion in hypothermia. J Physiol 206, 39, 1970.

60. Nelms, J. D. Adaptation of cold and cold injury. J R Nav Med Serv 58, 189, 1972.

61. O'Brien, J. R. Effect of cold in bleeding. Lancet 1, 89, 1971.

62. Otis, A. B. and Jude, J. Effect of body temperature on pulmonary gas exchange. Am J Physiol 188, 355, 1957.

63. Penrod, K. E. Cardiac oxygenation during severe hypothermia in the dog. Am J Physiol 164, 79, 1951.

64. Power, G. G., Aoki, V. S., Lawson, Jr., W. H. and Gregg, J. B. Diffusion characteristics of pulmonary blood gas barrier at low temperatures. J Appl Physiol 31, 438, 1921.

65. Raven, P. B. Niki, I., Dahms, T. E., and Horvath, S. M. Compensatory cardiovascular responses during an environmental cold stress, 5°C. J Appl Physiol 29, 417, 1970.

66. Rawlins, J. S. P. Thermal balance in divers. J R Nav Med Serv 58, 182, 1972.

67. Rogers, P. and Hillman, H. Increased recovery of anesthetized hypothermic rats induced by intra

carotid infusion. Nature 228, 1314, 1970.

68. Russell, J. C., McNeill, A. and Evonuk, E. Responses of the SCUBA diver to increased pressure and cold. Aerospace Med 43, 998, 1972.

69. Severinghaus, J. W. and Stupfel, M. Respiratory physiologic studies during hypothermia. The Physiology of Induced Hypothermia. Publication 451, National Academy of Sciences, National Research Council, Washington, D.C., 1956, Dripps, R. D., editor.

70. Severinghaus, J. W. Oxygemoglobin dissociation curve correction for temperature and pH variation in human blood. J Appl Physiol 12, 485, 1958.

71. St. Rose, J. E. M. and Sabistan, B. H. Effect of cold exposure on the immunologic response of rabbits to human serum albumin. J Immuno 107, 339, 1971.

72. Trevino, A., Razi, B. and Beller, B. Characteristic ECG of accidental hypothermia. Arch Intern Med 127, 470, 1971.

73. Vandam, L. D. and Burnup, T. K. Hypothermia. N Eng J Med 261, 546, 1959.

74. Webb, P. Calorimetry during treadmill exercise. Physiologist 12, 387, 1969.

75. Webb, P. Body heat loss in undersea gaseous environments. Aerospace Med 41, 1282, 1970.

76. Whitby, J. B. Treatment after exposure to cold. Lancet 1, 377, 1972.

77. Wilson, O., Hodner, P., Laurell, S. and Nosslin, B. Thyroid and adrenal response to acute cold exposure in man. J Appl Physiol 28, 543, 1970.

78. Yonce, Lloyd, R. The integration of the cardiovascular response to diving. Am Heart J 79, 1, 1970.

79. Severe Accidental Hypothermia, Editorial. Lancet 1, 237, 1972.

80. Emergency treatment of accidental hypothermia. Drug Ther Bull 9, 5, 1971.

81. Discussion following cold/wet survival symposium held on 4 & 5 November 1971. J R Nav Med Serv 59, 1, 1973.